Rugby Unions of less value in establishing the outcome of any changes in policy.[1]

I support the need for a register of injuries, but such a study has to be prospective and a standard form has to be produced. Someone in each club, whether it be a doctor, team captain, or physiotherapist, will have to take responsibility for filling up these forms, checking them, noting time off work and how the injuries were sustained, etc, and seeing that they are centrally registered. It is no good leaving it to the players to fill in forms as self notification is notoriously inaccurate, and, particularly with minor injuries, players may well not report to their general practitioners.

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1 Garraway WM, Macleod DAD, Sharp JCM. Rugby injuries: *BMJ* 1991;**303**:1082-3. (2 November.)
2 Silver JR. Injuries of the spine sustained in rugby. *BMJ* 1984;**288**:37-43.
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SIR,—In their editorial W M Garraway and colleagues highlight the problem of injury in rugby football and suggest the need for formal audit.[1] In fact, a study is currently under way.

Since 1985 the English Rugby Football Union has conducted a survey of injuries, involving all affiliated clubs and schools. At the beginning of each season copies of a detailed form relating to the nature and circumstances of injury are circulated and a request made for an officer from each organisation to be responsible for their completion and return. The data form the basis of an annual report that is available free from the English Rugby Union. This endeavour encompasses the aims outlined in the editorial and is likely to produce useful information.

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SIR,—I endorse the suggestions in W M Garraway *et al*'s editorial on rugby injuries that the rugby football unions establish a case register of injuries[1] but suggest that such a register be extended to cover minirugby. Minirugby (6-13 years) was set up to encourage the game in light of the demise of school rugby. It has been an overwhelming success.

The rules of minirugby are in a constant flux mainly because of the need to mitigate injury in such young players. The register could settle once and for all the most appropriate age to introduce the tackle and the hand off, and whether age or body weight should be the determining criterion when selecting a team. Although age is generally a good marker in younger boys, during the pubescent year quite remarkable weight and height differences can lead to unbalanced teams and consequent injuries. Further, those clubs that practise at the limits of the rules would be identified formally (we all know them). Paradoxically, this might allow some reasonable relaxation of rules designed to curb such clubs but often to the detriment of the natural rhythm of the game.

It is my experience, as an attending medical officer, that the number of injuries increases exponentially during competition matches. Intraclub matches rarely give rise to injury and I cannot recall an injury of note during training sessions. Interclub matches, however, always give rise to some injuries. I feel this is in part due to the often vociferous support from parents on the touchline, driving their boys to take unnecessary risks. The proposal in the editorial is overdue and would lead to a fall in the number of minirugby injuries which, although not great, must always be of concern.

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Chorionic villus sampling

SIR,—We find it remarkable that, at a time when the initial confusion over the safety and accuracy of chorionic villus sampling is being clarified by centres with large accumulated experience, Richard J Lilford suggests that the procedure should become history.[1] Provided that chorionic villus sampling is performed after 10 weeks in centres with experience, there is no increased risk of disturbance to embryogenesis and the rate of fetal loss is comparable with that associated with amniocentesis in the second trimester.[2-4]

Inaccuracy is almost entirely due to confined placental mosaicism,[5] which occurs in approximately 1% of cases (provided cytogenetic analysis is performed with both the direct preparation and culture).[6] (Mosaicism occurs with amniocentesis and can be of a similar order of magnitude.[7]) In more than four fifths of this 1% of cases[5] the fetus does not seem to be clinically affected as the effect of mosaicism depends on the chromosomes involved and the proportion of cells in the individual tissues.[8] Therefore it is possible for mosaicism to be diagnosed by chorionic villus sampling but not confirmed by amniocentesis or fetal blood sampling,[9] although this is believed to be rare. The implications for management are that termination should never be performed for mosaicism without further investigation and expert interpretation.

In conclusion, we consider that in centres with experience chorionic villus sampling has "risen" and should not be aborted.

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1 Lilford RJ. The rise and fall of chorionic villus sampling. *BMJ* 1991;**303**:936-7. (19 October.)
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SIR,—Richard J Lilford has always advocated decision analysis and frequently expounds on the question of choice. His editorial is subtitled "midtrimester amniocentesis is usually preferable"[1] and the inference from this—that first trimester chorionic villus sampling is passé—contradicts the idea of appropriate risk management, something that most practising clinicians appreciate.

The higher rate of fetal loss with villus sampling before 28 weeks' gestation reported in the European trial[2] was not substantiated by the Canadian study.[3] Lilford conceded that operator experience and expertise counts. The European trial in which 17% of procedures were considered difficult and 31% required more than one attempt to obtain adequate diagnostic material cannot suggest villus sampling is more risky than amniocentesis. Villus sampling, however, should be done by experts.

World cohort and personal experience of over 1000 samplings suggest that the rate of fetal loss with villus sampling is within 1-2% of the rate with amniocentesis (1·6% in the European trial[2]). Who should choose the screening procedure? Many mothers would not consider midtrimester amniocentesis preferable when faced with the emotive and physical cost of a midtrimester abortion.[4,5]

Facial clefting defects are common abnormalities[6] and are often associated with limb defects[7] in many syndromes. These defects are evident by the third or fourth week and established by the sixth week of gestation. The question of risk framing is important as many women seeking prenatal diagnosis may not consider oromandibular or limb hypogenesis a threat when the calculated incidence is 0·3 per 1000.[1]

The ambiguous results for mosaic chromosomal abnormalities reported in the Canadian trial were not a major problem in the European trial or the United States multicentre study of over 6000 women.[1] Clearly there is also a learning curve for cytogenetists and experience counts.[7,8] With regard to amniocentesis before 12 weeks' gestation, apart from the safety question, where are the amniotic fluid cells from?

Chorionic villus sampling was developed to meet women's needs to avoid midtrimester diagnosis and late abortion. As a member of the working party for the European trial I am acutely aware that participants were still on the learning curve and results will differ if the trial is repeated. Lilford must remember trials are conducted to provide answers and figures—the ingredients for risk framing and decision analysis. The choice must remain with the consumer, who may not be impressed by risk below statistical detection.

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Oral and intravenous rehydration therapy

SIR,—Angela Mackenzie and Graeme Barnes compared oral and intravenous rehydration therapy in children and came to the surprising but comforting conclusion that "rehydration by mouth or nasogastric tube is a safe and effective treatment in moderately dehydrated children with gastro-

enteritis."[1] The authors detail the oral therapy they used in terms of mmol/l.

Many years ago I was taught that the best oral rehydration solution was one that was made up of eight tablespoons of sugar and half a teaspoon of salt in one litre of water. Three glasses of this mixture were to be given after each stool. Does this simple, plain, and uncomplicated oral mixture, which could be understood by anyone, correspond to the glucose-electrolyte solution, measured in mmol/l, mentioned in the article?

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1 Mackenzie A, Barnes G. Randomised controlled trial comparing oral and intravenous rehydration therapy in children with diarrhoea. *BMJ* 1991;**303**:393-6. (17 August.)

AUTHORS' REPLY,—R K Edwards suggests making oral rehydration solution by adding eight tablespoons of sugar and half a teaspoon of salt to a litre of water; this would make a solution containing about 7% glucose (390 mmol/l) and sodium chloride 43 mmol/l. The glucose concentration of this solution would be dangerously high; glucose concentrations over 160 mmol/l (2·9%) are associated with less absorption of water and increased diarrhoea.[1] Most fruit juices and carbonated beverages (for example, lemonade, Coke, Pepsi, and Fanta) contain about 10% sugar,[1] so they should not be given undiluted to patients with diarrhoea. Edwards's recipe is probably a corruption of a World Health Organisation formula of eight level teaspoons of sugar and one level teaspoon of salt in a litre of water.[2]

For mild diarrhoea a suitable oral solution can be made at home by dissolving one heaped teaspoon of sugar (7 g sucrose, 3·5 g glucose) in a large cup of water. If the cup holds 200 ml this makes a solution containing about 1·8% glucose (100 mmol/l). The composition of home made solutions is very variable,[1] and we think that they should not contain salt because of the risk of salt poisoning. In areas with no cholera diarrhoeal stools usually have a low sodium concentration, and an appreciable sodium deficit is unlikely with mild diarrhoea (particularly if a solid diet is continued). For severe diarrhoea, particularly if there is dehydration, prepacked correctly formulated salts should be used to make a solution containing sodium 50-90 mmol/l, potassium 20-30 mmol/l, chloride 40-80 mmol/l, citrate 10 mmol/l, and glucose 83-111 mmol/l (1·5-2%).

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Auditing necropsies

SIR,—I Lauder's editorial[1] on the joint working party report *The Autopsy and Audit*[2] rightly concludes that necropsy is an excellent method of audit and needs to be encouraged. The report expresses concern about the fall in hospital necropsies, and Lauder suggests that this is because junior doctors find them distasteful and do not realise their value.

The report also suggests that the responsibility for obtaining a necropsy should lie with the consultant in charge of the case. The working party presumably think that this will result in more necropsies being performed, but I think they have misinterpreted the problem. Many consultants already instruct their junior staff to request a necropsy in all cases, unless the cause of death is in no doubt. Unfortunately, permission from the relatives is still refused more often than it is given, even when the consultant is directly involved. A similar situation exists with requesting organs for donation, and it seems to me that the general public, although becoming better informed on medical issues, is becoming increasingly squeamish about having its deceased relatives "cut up." How often have we heard the phrase, "He or she has been through enough already?" This phrase is often used in cases where there have been complications, and although the final cause of death may be clear much information can be gained from a necropsy that might improve our management in the future. Unfortunately, the argument of medical education is usually ignored by relatives.

Another problem is the reluctance of certain coroners and coroner's officers to request a necropsy when a case is reported to them, and I am sure Lauder is aware of this. One possible explanation is that the coroners are also trying to save money, and perhaps they too need to be educated about the educational value of a necropsy. Unfortunately, it is unlikely that district medical audit advisory committees will have any influence over them.

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1 Lauder I. Auditing necropies. *BMJ* 1991;**303**:1214-5. (16 November.)
2 Joint Working Party of the Royal College of Pathologists, Royal College of Physicians of London, and the Royal College of Surgeons of England. *The autopsy and audit.* London: RCPath, RCP, RCS, 1991.

Communicating necropsy results

SIR,—The findings of Paula Whitty and colleagues on the communication of necropsy results in North East Thames region cannot be accepted as they stand.[1] Although a delay in receiving the final report of 144 days is clearly unacceptable, 22 days may be a good performance, depending on the case mix at the particular mortuary. For example, many neurology cases require fixation of the brain for several weeks before even the macroscopic findings are available, let alone the histological results.

The authors do not mention how often members of the clinical team attended the necropsy. The doctor has to know the cause of death before requesting a hospital postmortem examination. The usual reason for a request is clinical interest, and it is therefore common for one or more of the clinical team to attend. Many pathologists would not consider it necessary to send out a preliminary report when the findings have been witnessed by members of the requesting firm.

The authors also imply that postmortem histology is somehow an optional extra rather than an integral part of the examination. I consider that a postmortem examination is as much an intellectual process as a physical one. It should start with a consideration of the history and should finish with the drawing of conclusions after consideration of the macroscopic findings, histological findings, and results of any microbiological or toxicological tests considered necessary. I am sure that the average pathologist's macroscopic diagnosis on a colectomy specimen is at least as accurate as most postmortem diagnoses, but would you expect a surgeon to be satisfied with a macroscopic diagnosis alone? The answer is not to try and restrict postmortem histological examination but to make sure it is done without unnecessary delays—something that many laboratories manage as a matter of course.

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Corticosteroids and tuberculosis

SIR,—Martin B Allen and Nigel J Cooke point out that lymph nodes may enlarge during antituberculous chemotherapy.[1] But lymph nodes may also enlarge in patients who have successfully completed chemotherapy.[2]

A 30 year old man started standard chemotherapy for cervical and supraclavicular tuberculosis adenitis (fully sensitive organisms). The lymph nodes decreased in size but after six months enlarged again. One was incised, but no acid fast bacilli were grown. Five months after he had finished 18 months' chemotherapy the lymph nodes again enlarged and became extremely uncomfortable. The lymphadenopathy resolved after two weeks' treatment with 20 mg prednisolone daily. He had no further problems in the subsequent two years.

It has been postulated that the mechanism for the enlargement of sterile nodes is a reaction to tuberculoprotein.[2] The enlargement is usually transient, but occasionally treatment with steroids may be necessary.[2]

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1 Allen MB, Cooke NJ. Corticosteroids and tuberculosis. *BMJ* 1991;**303**:871-2. (12 October.)
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Health of the nation

SIR,—Peter Anderson's article on alcohol and the health of the nation cannot be allowed to pass without comment.[1] From 1981 to 1982 alcohol consumption fell from 8·95 to 8·76 litres of pure alcohol per adult, not from 10·4 to 9·2 litres.[2] Convictions for drunkenness fell by 2%, not 11%, and drink-driving convictions increased by 3%—they did not fall by 8%.[3]

Anderson quotes a paper by Kendell *et al* in support of his belief that a substantial increase in the price of alcohol would affect heavy drinkers and more modest consumers alike.[4] In that paper only one factor (changes in excise) was selected as affecting patterns of consumption during 1978-81. Others could have been important—for example, employment and changes in income. Many of the heavy drinkers whose consumption declined during 1978-81 had become unemployed. Interestingly, too, during this period some light drinkers became heavy drinkers—a rather perverse result of changes in excise.

Anderson argues for a reduction in the population mean, which, in his opinion, predicts the number of heavy drinkers. But it is just as likely that the number of heavy drinkers predicts the population mean.

The claim that a 1% decrease in licensed outlets would result in a 2% decrease in consumption is contradicted by recent experience in the United Kingdom. During 1979-89 the number of licensed premises increased by 16% (from 170 100 to